proinflammatory cytokine linked to a PD1 ligand. The ligand specifically binds to a receptor on the immune cell thereby activating the cell.

[0015] In still yet another aspect, the disclosure provides a method to treat a tumor comprising identifying a subject with a tumor and administering to the subject a therapeutically effective amount of a composition comprising a proinflammatory cytokine linked to a PD1 ligand.

[0016] In a different aspect, the disclosure provides a method to treat a viral infection comprising administering to the subject a therapeutically effective amount of a composition comprising a proinflammatory cytokine linked to a PD1 ligand. In other aspects, the disclosure provides a chimeric peptide comprising a cytokine peptide and a PD1 ligand peptide.

[0017] In certain aspects, the disclosure provides a chimeric peptide comprising a cytokine peptide and an anti-PD1 antibody.

[0018] In another different aspect, the disclosure provides a nucleic acid molecule comprising a sequence encoding a chimeric peptide of the disclosure.

[0019] In yet another different aspect, the disclosure provides a pharmaceutical composition comprising a chimeric peptide of the disclosure.

[0020] In still yet another different aspect, the disclosure provides a method of treating a subject diagnosed with cancer comprising administering to the subject a pharmaceutical composition of the disclosure.

[0021] In another aspect is a method to treat a tumor by (1) identifying a subject with a tumor; and (2) administering to the subject a therapeutically effective amount of a combination therapy described herein.

[0022] In another aspect is a method for treating a viral infection, by administering to the subject a therapeutically effective amount of a combination therapy described herein.

[0023] In some embodiments of the various methods provided herein, a pharmaceutical composition of the disclosure is administered in combination with a PD-1 inhibitor. In certain embodiments, the PD-1 inhibitor is an anti-PD-1 antibody. In some embodiments, the anti-PD-1 antibody is an antagonistic antibody. In some embodiments, the PD-1 inhibitor is selected from the group consisting of nivolumab, pembrolizumab, pidilizumab, REGN2810, PDR 001, and MEDI0680.

[0024] In other embodiments, of the various methods provided herein, a pharmaceutical composition of the disclosure is administered in combination with a PD-L1 inhibitor. In certain embodiments, the PD-L1 inhibitor is an anti-PD-L1 antibody. In some embodiments, the anti-PD-L1 antibody is an antagonistic antibody. In some embodiments, the PD-L1 inhibitor is selected from the group consisting of durvalumab, avelumab, atezolizumab, or BMS-936559, STI-A1010, STI-A1011, STI-A1012, STI-A1013, STI-A1014, and STI-A1015.

BRIEF DESCRIPTION OF THE FIGURES

[0025] The application file contains at least one drawing executed in color. Copies of this patent application publication with color drawing(s) will be provided by the Office upon request and payment of the necessary fee.

[0026] FIG. **1**A, FIG. **1**B, FIG. **1**C, FIG. **1**D, FIG. **1**E and FIG. **1**F depict a diagram, immunoblot and graphs showing the generation and in vitro evaluation of OMCP-mutIL2. (FIG. **1**A) Schematic structure of OMCP-mutIL2. (FIG. **1**B) Molecular weight of OMCP-mutIL2 compared to mutIL2 and wild-type IL2. IL2, mutIL2, and OMCP-mutIL2 were produced in mammalian cells and have higher molecular weights due to glycosylation. The lower migrating band for mutIL2 corresponds to unglycosylated protein, likely due to lysis of the producing cells. Based on differences in molecular weight all cytokines and construct were administered on a molar basis with 1 μl of 4.4 μM solution defined as 1000 IU equivalents (IUe) herein. This effectively allows for equimolar comparison between IL2, mutIL2 and OMCP-mutIL2 despite different molecular weights. (FIG. **1**C, FIG. **1**D) In vitro activation of NJ lymphocyte subsets after 36 hours of culture in 100 IUe of cytokines or OMCP-mutIL2 construct. (FIG. **1**E, FIG. **1**F) Proliferation of B6 lymphocyte subsets after 5-day culture in 1000 IUe/ml of cytokines or OMCP-mutIL2 construct. Graphs representative of 3-6 replicates per condition. black=saline; blue=wtIL2, red=OMCP-mutIL2, green=mutIL2.

[0027] FIG. **2**A, FIG. **2**B, FIG. **2**C, FIG. **2**D, FIG. **2**E, FIG. **2**F, FIG. **2**G, FIG. **2**H, FIG. **2**I, FIG. **2**J, FIG. **2**K, FIG. **2**L, FIG. **2**M, FIG. **2**N and FIG. **2**O depict graphs and images showing in vivo dosing of IL2 and IL2 constructs. Animal mortality (FIG. **2**A) and morbidity assessed by weight loss (FIG. **2**B) accumulation of ascites and pleural fluid (representative syringe-FIG. **2**C; average from all mice in the group-FIG. **2**D) and (FIG. **2**E) organ inflammation after administration of wtIL2. Animal mortality (FIG. **2**F, FIG. **2**H, FIG. **2**J) and morbidity as assessed by weight loss (FIG. **2**G, FIG. **2**I, FIG. **2**K) after administration of high dose wtIL2 (FIG. **2**F, FIG. **2**G), OMCP-mutIL2 (FIG. **2**H, FIG. **2**I) and mutIL2 (FIG. **2**J, FIG. **2**K) in anti-AsialoGM1 (solid line) or rabbit IgG-treated (dotted line) in A/J mice. Weight loss (FIG. **2**L), ascites (representative syringe-FIG. **2**M; average from all mice in the group-FIG. **2**N) and organ inflammation (FIG. **2**O) in mice treated with 200,000 IUe of either wt IL2, OMCP-mutIL2 or mutIL2. All graphs represent 46 animals per treatment condition. ns $p>0.05$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$; black=saline; blue=wtIL2, red=OMCP-mutIL2, green=mutIL2.

[0028] FIG. **3**A, FIG. **3**B, FIG. **3**C, FIG. **3**D, FIG. **3**E, FIG. **3**F, FIG. **3**G, FIG. **3**H, FIG. **3**I and FIG. **3**J depict graphs and images showing immunologic changes associated with IL2 and IL2 construct administration in vivo. (FIG. **3**A, FIG. **3**B) Total splenocyte counts after a five-day course of 200,000 IUe of IL2 (blue), mutIL2 (green) and OMCP-mutIL2 (red). (FIG. **3**C) NK cell expansion and activation after IL2, mutIL2, OMCP-mutIL2, high dose IL2, high dose mutIL2 and IL2/anti-IL2 complexes measured by cell counts in the spleen (top) and KLRG1 upregulation (bottom). (FIG. **3**D) $CD4^+Foxp3^+$ T_{reg} expansion and activation as measured by cell counts in the spleen (top) and ICOS upregulation (bottom) as well as (FIG. **3**E) NK/T_{reg} ratio in the spleen. Expansion of splenocytes (FIG. **3**F, FIG. **3**G) and NK cells (FIG. **3**H) in B6 mice treated with 750,000 IUe of cytokine or construct. T_{reg} expansion and activation (FIG. **3**I) as well as $NK:T_{reg}$ ratio (FIG. **3**J) in the spleen of B6 mice. All graphs represent an average cell count±SEM from 5-10 mice per group. ns $p>0.05$; * $p<0.05$; ** $p<0.01$; *** $p<0.001$; black=saline; blue=wtIL2, red=OMCP-mutIL2, green=mutIL2.

[0029] FIG. **4**A, FIG. **4**B, FIG. **4**C, FIG. **4**D and FIG. **4**E depict graphs and images showing cytokine-mediated tumor immunotherapy. (FIG. **4**A) In vivo cytotoxicity for YAC-1 lymphoma after intravenous injection. (FIG. **4**B, FIG. **4**C)